THE

PATHOLOGY

OF

BRIGHT'S DISEASE,

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WITH ILLUSTRATIONS.

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THE PATHOLOGY OF BRIGHT'S DISEASE.

The most serious obstacle to the advance of medical science in all time has been an inveterate tendency on the part of its votaries to theorise rather than to observe. This habit was ancient in the days of the father of medicine, and even now, in this practical age, it sways a powerful, though we trust a diminishing influence. The very sequence of terms in the title, *Professor of the Theory and Practice of Medicine*, is an outgrowth as well as an index of this state of the medical mind. John Watson, when writing of the great importance attached by Hippocrates and his followers, to prognostic indications, remarks that as a consequence—

"The individual types of disease were not as thoroughly investigated as they might have been. Had they dwelt on these with greater care, it is possible that most of the diseases which are now looked upon as of comparatively recent origin, and for accounts of which we search in vain among the ancient medical authorities, might be shown to have existed from the earliest times."* Thus it followed that "the descriptions of epidemic and other diseases by the early historians, who drew from observation only, who made no pretensions to medical knowledge, and whose minds were unembarrassed by the training of the schools, are at the present day more worthy of reliance, than the accounts rendered of the same diseases by contemporary medical authorities."† So prolonged and baneful was the influence of this

* The Medical Profession in Ancient Times: page 60.
† Ibid. page 61.

philosophic spirit, that in 1586, we find Thomas Gale, Master in Surgery, defining thus a most vital department of our science. "Phisiologia is that which doth consider the VII naturall thinges whereof the body of man is made, as Elements, Temperaments, Humors, Members, Spirites, Virtues, and Operations. The Elements be foure, as Fier, Ayer, Water, and Earth. The Humors be foure, also: as Bloud, Choller, Flegme, Melancholly. And the Temperaments be foure likewise, as Hot, Cold, Moyst, and Drye. These foure are the matter whereby all the members of the bodie are made with, the temperaments, and spirites, therein contained."* From the abandonment of the peculiar views of the school of Cnidos, which was eminently a school of observation, until about 1770, renal pathology seems to have slumbered, or to have become extinct. The kidneys were not suspected of disease, but relied upon confidently to mirror in their secretion the condition of various internal organs.

More than a century earlier than the above date, we find Hamand writing thus of the pulse and urine.

"The *Pulse*, then, or *venæ pulsatiles*, pulse veins, are signals to declare the state of the heart and arteries, the urine of the liver and veins, and which of them is of most certaintie, *quaeritur*. * * * Urine in waveing itself with the bloud, conveyeth itself into all parts of the body; and from thence back again into the veins, liver, and the vessels of the *Urine* retaineth with it some *Symptoms* of the parts affected from whence it proceedeth."† And again; "Urine being unprofitable excrement, descendeth into the Ureters or water-conduits, and from thence into the bladder; where, by the assistance of the muscle Sphincter, which is *musculus-*

* Certaine Workes of Galen's, Thos. Gale, Lond. 1586, page 24.

† Ourography, or Speculations on the excrements of Urine: Henry Hamand, Lond. 1656, page 2.

constrictivus shutting the neck of it, it is at man's pleasure in part voyded, but not all; for some part thereof together with the bloud, is distributed into every member of the body and voyded afterwards: and therefore urine is not only made of that watery substance which is drawn from the Liver; but also from the greater and lesser veins, and from the whole body.* * *So also it doth reveale to the judgment of exact inspection, the griefs and maladies residing in the smaller veins, and substance of the whole body."*

During the third quarter of the last century, two writers appeared, who, dissatisfied with the crude doctrines which then obtained in regard to dropsy, sought in some abnormal state of the internal organs, a more satisfactory explanation of certain cases of that disease.

Cotunius in Naples, and Hewson in London, about the same time, were the first to hint that dropsy was not a substantive pathological condition. They did not however attribute its causation to disease of any particular viscus. In England at least, an albuminous state of the urine was not generally recognized until about the year 1813, when the work of Dr. Blackall called attention to the fact that in a large proportion of dropsical patients, this secretion coagulated upon the application of "heat or nitrous acid." Thus was constituted the new class of inflammatory dropsies. A dozen years later, Dr. Richard Bright was enabled to trace these cases to disease of the kidneys as their cause, and so gave to rational pathology an impulse, which half a century has done nothing to abate.

This celebrated author pointed out three or four forms of renal disease associated with albuminuric dropsy, but left to others the investigation of the structural changes, peculiar to each, while he devoted himself to the study of their clinical

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* Ibid, pages 6—7.

history. The field thus opened was speedily occupied by earnest workers in every part of Europe: Christison and Gregory of Edinburgh, Osborne of Dublin, Rayer and Solon of France, Forget of Strasburgh, Frerichs of Brunswick, Traube, Virchow, Johnson, and Dickinson, together with many others whom they represent, are distinguished by their contributions to the science of Pathology both general and special. Our branch has not, however, advanced with regular and graduated steps from its inception to the comparatively mature development of the present. Renal literature discloses a vast amount of darkness which is not yet totally dissipated, much of which has been engendered by the very students themselves.

We must thank Virchow for the classification of kidney diseases which is now generally adopted by English writers. In his "Cellular Pathology" he shows that the three principal anatomical elements of renal structure are each in turn subject to a characteristic morbid change, and that we may thus have an organic lesion affecting primarily the tubes and their lining, another producing at first increase and afterwards contraction of the fibrous stroma; and a third attacking the vasular supply of the parts.*

If we accept a three-fold division as the first law of renal pathology, the second is that one of these types of disease may excite another, so that it is not unusual to find two, or, apparently, even all of them co-existing in the same organ.

Albuminuria is a broader term than "Bright's Diseases," as Dr. Stewart puts it. Albumen may be found in the urine of those who are neither suffering from admixture of blood or pus with that fluid, nor yet from seated organic lesion of the kidney, and yet in whom this appearance cannot be termed physiological with any degree of propriety.

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*Cellular Pathology, Phila. 1863, pp. 422—425.

The only visible change is great congestion, and under the microscope, a slight and not necessarily general cloudiness of the epithelium. The renal derivatives discovered in the urine are precisely similar to such as occur temporarily in many congestive disorders from which complete recovery is the rule. Dickinson claims, we think with propriety, that oft-repeated, and long continued venous, or passive congestion, is apt to result in that form of nephritic disease in which the fibrous matrix is primarily involved. He therefore describes that condition under the head of granular degeneration. Active congestion, or that produced by irritant drugs, and various diseases, he classes with tubal nephritis, and adds, we think without sufficient ground, "Even in the mildest cases, the urine contains the characteristic casts and epithelial deposit, and when there occurs an opportunity for examination of the kidney, the tubes are found to be obstructed with epithelial growth."* But although these two forms of congestion are at times related to the respective morbid conditions above cited, it is nevertheless true that in a vast majority of instances the more serious lesion never follows the more trivial. Many a patient who has succumbed to diphtheria or erysipelas, has been found shortly before or after death, to have had more or less marked albuminuria, but we think that it would be manifestly improper to claim upon such evidence alone, that these individuals had died of kidney disease.

On such practical grounds we think that Dr. Wm. Roberts† and other authors, are justified in classing renal congestion as a separate morbid state, included in the term Albuminuria, but not in Bright's Disease. For the same reasons we cannot approve Dr. G. Stewart's selection of cases to illustrate

*Pathology and Treatment of Albuminuria, p. 83.

†Urinary and Renal diseases, p. 273.

"acute nephritis terminating fatally in the first stage."* In the first two of these the renal lesion was probably nothing more serious than congestion, and had little or nothing to do with the causation of death.

The following cases may serve as illustrations of this condition.

I. A fine male infant of fourteen weeks died at Charity Hospital. The symptoms were obscure, except that it was comatose at the end. The mother had typhus fever, and the child nursed until within a week of its death. At the autopsy no special lesions were discovered, save deep congestion of the liver and kidneys. The bladder was distended with about one fluid ounce of limpid urine, which however, was found to be largely albuminous, and to contain granular casts with much epithelium, from all parts of the urinary tract. These conditions were suggestive of acute nephritis, and to solve the doubt, a portion of one kidney was hardened in a solution of bichromate of potassa with a view to subsequent microscopical examination. After the proper degree of firmness had been attained, a thin section was colored in carmine, rendered transparent by turpentine, and preserved in canada balsam. The following wood-cut represents the condition in which the tubes and their cellular lining were found.

With regard to the preparation of the specimens from which the illustrations of this paper have been traced, it may be observed that they were all subjected to the action of chromic acid or its potassa salt. Some of them were then put up permanently in camphorated glycerine, without further treatment, while others were colored and eventually mounted in balsam as above stated. The latter process is fully de-

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* Bright's Diseases of the Kidneys, pp. 21—22.

scribed by Dickinson,* and in several works upon the use of the microscope.

Section of congested kidney from an infant fourteen weeks of age, showing tubules and malpighian body in a nearly healthy condition. 220 diameters.

In making the above section, the knife passed directly through the cortex toward the pelvis, and the portion here represented lies within one line of the capsule. The cut shows a group of malpighian bodies, only one of them fully drawn, and several uriniferous tubules intersected at various angles. The epithelium of the tubules is seen to be almost perfect. The nuclei are visible in nearly every instance. The cells are perhaps slightly more granular and dark than is normal, and in one or two tubes a few of them are seen to have become detached from the basement membrane. This last, however, is a very common observation in normal

* Vide Opus citat. pp. 12-13.

kidneys. All of the tubules are quite pervious. In altering the adjustment, the cells were found to be sufficiently transparent, the nuclei of those out of focus being shadowed in a manner not perceived, when the epithelium has become decidedly cloudy. In another part of the same section the minute blood vessels were seen to be densely crowded with blood corpuscles. No ruptures were to be found. The microscope, then, completely failed to confirm the conjecture that an inflammatory stage had been reached.

II. Less than one drachm of milky urine was pressed from the bladder of A. S., seven months, at the autopsy. Death was caused by convulsions due to intestinal irritation. There was some rigidity of the muscles, and the thumbs and great toes were adducted and flexed. Lungs congested, with points of emphysema in both, and a lobule of pneumonia in the right apex. Heart firmly contracted, containing small red and whitish clots. All of Peyer's patches were enlarged, red, raised, and roughened to the feel. The kidneys did not appear congested. There were some dark lines, but as a whole they were considered normal *upon inspection.* In a filtered portion of the urine a coagulum was produced by heat and nitric acid. Its turbidity was found to be due to epithelium, as had been supposed. The cells were of all varieties and sizes found in the urinary tract, and were probably in large part removed from their attachments by the pressure of the hand in evacuating the bladder. Numerous blood corpuscles were present, more or less swollen and colorless; vibriones also and minute spore-like bodies. A few casts were found which contained blood corpuscles, or granular matter; others were hyaline. They were for the most part quite small.

A portion of one these kidneys was also submitted to microscopical examination after hardening. This section was taken from a position somewhat more remote fro m the

capsule than in the previous instance. No malpighian tufts are here shown. Certain open spaces appear, which are due to the forcible removal of the tube contents in the process of cutting and mounting the specimen. The view is taken just at the edge of the section where the parts happened to be thinnest, and most favorable for tracing. These spaces are not found in the more central parts. The only abnormal conditions noticed were slight general cloudiness of the epithelium, and the partial filling of tubules here and there with granular debris. The latter, like detachments of epithelial cells, is not necessarily an indication of pre-existing renal disease. It is produced by the breaking down of cells thus loosened and thrown off.

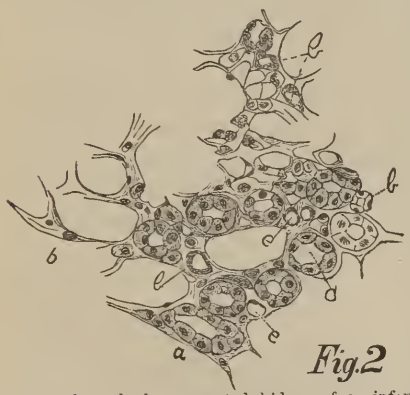

Fig. 2

Section through the congested kidney of an infant seven months of age. 200 diameters.

The wood-cut represents all the tubular structures of the kidney cut nearly transversely. The epithelium is seen to be in place, transparent though somewhat cloudy, and scarcely if at all swollen. At *a* is seen a tubule cut obliquely. At *c* is represented the convoluted tubule in its condition after leaving the malpighian tuft, the descending arm of the loop of Henle. The ascending reach is shown at *b*, recognized by the smaller size of the tube and flattened epithelium. The nuclei of the fibrous stroma, and, perhaps, sometimes, the capillaries, are shown at *d*, while at *e* are small blood vessels.

Here, then, are two instances, which certainly come

within the limits stated by Dr. Dickinson, (as previously quoted), and would probably be classed by that author under the head of tubal nephritis, the inflammation being supposed incipient, if not fully developed. And yet the microscope fails to detect any indication of inflammatory action, notwithstanding the presence of albumen, casts, and even blood in the urine. What evidence have we, that these patients, had they passed the acme of their respective maladies, as multitudes constantly do, would not quickly have lost all traces of renal disturbance? Surely nothing in the condition of the organs in question could militate against such a supposition. It must doubtless be admitted that, had these cases proceeded to the stage of inflammation, they would have assumed the tubal form of disease; in the same manner it is granted that the organic lesion following venous congestion is of the granular type, but these concessions do not require the admission that all active and all passive congestions are necessarily to any extent either tubal or granular diseases of the kidney.*

The conclusion is thus reached that in addition to "Bright's Diseases," renal congestion, active or passive, should retain a separate position in our nosology. The sections from which these illustrations have been traced are in the possession of the author, and may be inspected by any who are interested in this subject.

The three types of morbid processes properly included in the term Bright's Disease are as follows:

I. Tubal nephritis, also called *acute desquamative nephritis*,

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* Cornil refers to the very frequent appearance of albumen in the urine found in the bladders of corpses as due to a post-mortem change. He quotes Gubler as holding the same opinion. (*Des Différentes Espèces de Néphrites, Paris*, 1869, p. 21 note.) In the above cases, however, the presence of casts, and even of blood, gave evidence of ante-mortem congestion.

acute diffuse nephritis, the inflammatory form of Bright's Disease, croupous nephritis, and acute Bright's Disease.

II. Granular degeneration, also called *chronic desquamative nephritis, parenchymatous nephritis, the cirrhoticor contracting form of Bright's Disease, the gouty, or the fibroid kidney.*

III. The waxy, *depurative, amyloid or lardaceous* disease.

Authors who make the duration of the malady a basis for classification, include the second and third division under the term *chronic Bright's Disease.*

It has often been questioned whether the *acute stage* of tubal nephritis should be called by the name of Bright. There are, however, cogent reasons for its description in the connection above indicated. Prominent among these is the fact, that, although numerous cases of tubal disease run an acute course and quickly terminate in recovery or death, there are too many which become chronic, and during life, at least, are liable to be confounded with the other forms. Again, it is highly probable that Bright actually described the kidneys of advancing chronic tubal disease in his second class, and that it is thus fully entitled to the rank given it by later authors. There would be manifest impropriety in treating separately processes which are known to be so closely related.

In tubal nephritis we find intense congestion attended with very active proliferation and desquamation of the epithelial lining of the tubules. The material thus thrown off blocks up and distends these canals, and effectually prevents the elaboration of urine. The congestion produces rupture of the ultimate arterial tufts and capillaries, and the tubules are then found to contain blood in addition to epithelium. The interrupted circulation, moreover causes an effusion of fibrin, or fibrinoid material which solidifies in, and takes the form of these canals, imbedding and agglutinating their contents, and constitutes in "casts" a most

efficient means of diagnosis. The kidney is now found to be increased in bulk, tense, and smooth, and the cut surface is at once bathed in blood. In cases which result from scarlatina, the catarrhal element is in excess of the congestive. In these, therefore, the kidney is less red and bloody, presenting on section a surface mottled finely, whitish and red, the epithelium producing the lighter color. At a later period, the tubular contents are seen to have lost their definite form and to have become opaquely granular, or disintegrated, forming an amorphous mass. This stage is easily traced in the casts, which now become more and more filled with debris, instead of well formed epithelial elements.

Should the disease still persist, the cells begin to undergo fatty change. At first but few betray this advance, but as it becomes more general, the casts may become laden with minute oil globules, not confined to individual cell limits. This constitutes the stage of fatty degeneration. It is a legitimate consequence of depraved cell growth, and may make its appearance in any form of renal lesion in which protracted interference with the normal condition of the tubes obtains. It may thus follow either granular or waxy change, but it is not apt to appear until the later stages of these affections. An organ in which fatty degeneration is beginning to develop, still has the mottled appearance characteristic of an earlier period, but there is less redness, and the light colored points begin to have a whiter, or at times, a light yellowish tinge. Later still, as the kidney becomes more completely invaded, the section is found uniformly white or whitish, and its markings are exceedingly indistinct. This is the "large white kidney." It probably never exists without having been preceded by an attack of acute tubal nephritis. Thin sections of an organ in this condition are very opaque, and show little else than oil globules and the fibrous matrix. This condition is an almost invariable con-

equence of chronic tubal nephritis. It is then in close pathological relationship to inflammation of the renal epithelium. The granular and waxy diseases it does not follow with any such regularity.

The following case, reported by house physician Jas. L. Robertson, illustrates the power of alcohol to excite tubal nephritis.

III. G. W., fifty-three years of age, a laborer, born in the United States, was admitted to Charity Hospital, Feb. 3rd, 1869. He stated that he had been drinking, and had not slept for two months. His entire person was covered with an eruption, presenting large, square, somewhat adherent scales, especially abundant over the extensor surfaces, and accompanied with considerable thickening of the integumentary tissues. He became delirious on the following day, after passing a sleepless night. The urine was now found to be bloody and highly albuminous; lower extremities œdematous; pulse strong, ninety beats to the minute. During the night of the 5th, he tried to jump out of the window, and it was found necessary to confine him. In the morning, although he had not slept, his appetite was very good, and he felt well; but he was very restless and had a dry tongue. On the 7th there was a slight amendment, the tongue and skin became more moist, and a little sleep was obtained. The urine however, was still scanty and high colored. On the 8th, his tongue was again dry. He could not be kept in bed, and was altogether in a worse condition than at any time since admission. February 10th, sinking fast; could not protrude his tongue; teeth covered with sordes; pulse could not be counted. Death occurred during the night. Autopsy thirty-two hours after death. The body was quite rigid, and covered with the eruption above described. The lungs were normal, except a slight trace of pneumonia in the lower lobe of the right. The heart was

hypertrophied, weighing 14½ ounces, but without valvular lesions. The liver, 58 ounces, was apparently normal. Spleen 6 ounces, soft. The kidneys, together 15½ ounces, were of a very dark color, extremely congested. The capsule was easily removed, showing a surface mottled with light colored points and lines. The same was noticed upon section. The cortical portion was thickened.

These organs were very fine specimens, and presented the disease in an early stage. Hardened sections of the cortex were mounted in glycerin and examined microscopically. The next three illustrations give an idea of the condition of the various elements involved.

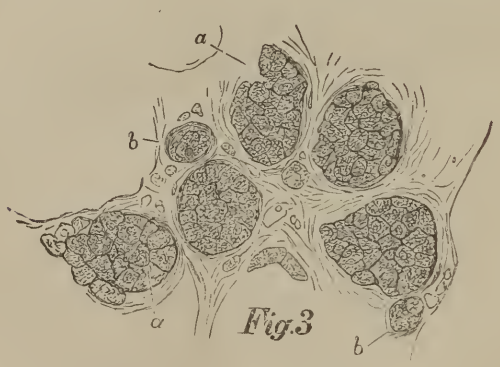
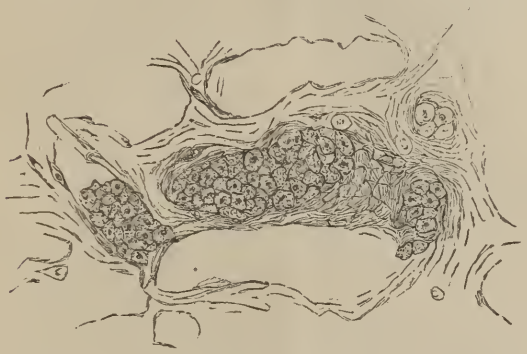


Fig. 3

Tubal nephritis; the tubules in transverse section shown densely filled with cloudy epithelium.—200 diameters.

Here are seen five of the larger convoluted tubules and three of the ascending tubes, all filled with granular, nearly opaque, swollen epithelium. In some parts, as at *a*, the cells are torn from their attachment to the basement membrane. Everywhere they are irregular in size, and in only a few instances are sufficiently transparent to allow their nuclei to

be seen. The smaller tube *b*, to the left, was apparently occupied by a fibrinous cylinder, in which a concentric arrangement of lines was noticed. No epithelium lined its walls, and but one or two cells could be detected in the contents. The tube *b*, to the right, is choked by the enlargement of its epithelium. Several nuclei and sections of capillaries are seen in the stroma.



Tubal nephritis; the tubules in longitudinal or oblique section. 200 diameters.

This cut is from the same section as the preceding, and shows that the diseased condition is not confined to certain points in the continuity of the tubules, but occludes them through considerable reaches. This representation is taken from a thin edge, and some of the cellular plugs are seen to have been displaced in mounting. The spaces do not appear in less disturbed parts. They are not due to the action of disease. In both of these illustrations, the fibrous material is seen to be relatively somewhat increased. It is highly probable that this slight hypertrophy is due to the prolonged irritation of alcohol taken in previous debauches.

Tubal nephritis; tubules filled with blood-corpuscles. 200 diameters.

Examination of the sections from which these tracings were taken, with a low power, made it evident that the filling of tubes with epithelium, though general, was not universal. Individual tubes here and there, or groups of a few, or even larger spaces would exhibit effusion of blood rather than cell proliferation.

Thus in the cut are seen five large tubules, in more or less oblique section, with a smaller ascending branch in their midst. The latter alone is choked with epithelium, while the former, and others in the immediate vicinity, contain blood corpuscles almost exclusively. These tubules for the most part retain their lining. There were points so covered with blood corpuscles that it was impossible to determine whether the cells were in place or not. In these tubes the rupture of the malpighian tufts had probably relieved the congestion sufficiently to prevent temporarily the inordinate development of cells. Those of the latter which still retained their position were nearly opaque: the nuclei were only occasionally to be seen.

Other tubules contained mingled blood and epithelium.

Granular degeneration of the kidney is probably always associated in its inception with long continued, active or passive congestion, particularly the latter, but it is not ushered in by any acute symptoms. The organ is at first

increased in bulk, and under continued morbid stimulus the cellular elements of the fibrous stroma become active and go through the changes involved in reproduction. This increase of the fibrous matrix, although very considerable in some parts, especially about the minute arterial tufts and branches, does not at first necessarily interfere much with the function of the organ. But contraction of the new material at length begins to occur, and in the course of years proceeds to such an extent, that instead of being increased in size, the kidneys are reduced occasionally to one-half or one third their normal weight. The capsule tears off with difficulty, and leaves a roughened and irregular surface. During this second stage, all the conditions are present which favor the lighting up of epithelial inflammation within the tubules. Indeed this very generally occurs once or oftener in the course of such a case. When uræmia appears in the granular disease it is probably due to this complication. Fatty degeneration of the epithelium may also result from this protracted interference, and it is in this way that the "small white kidney" is produced. Were it not for certain lesions of other organs which very commonly attend granular degeneration of the kidneys, but are never necessarily connected with acute nephritis, it would be difficult in such cases to decide which was cause and which effect, the granular or the tubal disease. But with the clinical history, and disease of other parts, especially the heart and liver to help us, there can be no hesitation under ordinary circumstances. We think that Stewart* and Cornil† have been led into error in describing a stage of atrophy as following inflammatory disease. All of the cases given by the former author in illustration of this state, were probably instances of the tubal lesion consequent upon granular degeneration.

* Bright's Diseases, p. 30–32.

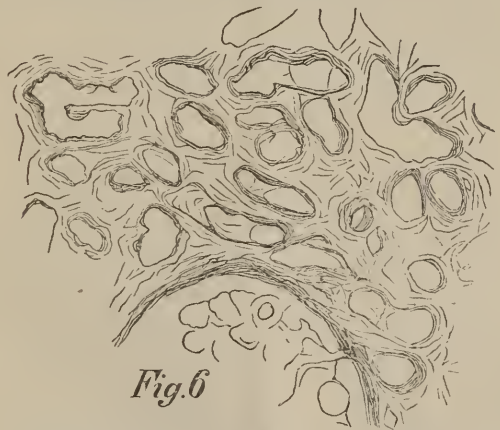
† Des Différentes Espèces de Nephrites, p. 49.

The contraction of newly formed fibrous cells is likely to take place very irregularly, and to constrict uriniferous tubules at various parts of their course. Such strictures are followed by dilatation above and contraction below the affected point, precisely such as occurs not infrequently in the ureters or urethra from various causes. The dilated portion may never become large enough to be visible to the naked eye, and a kidney may be filled with such microscopic "cysts," or they may attain a considerable size, from that of a pin head to that of an orange, and exist in such small numbers, as not seriously to interfere with the functions of the organs, or in such multitudes as to involve the total destruction of the glandular structure. In this condition the organ may attain many times its normal weight and size, but is nothing more than a congeries of closed sacs filled with fluid.

The term "granular" needs some explanation. As originally used by Bright, the expression relates to a more or less distinct mottling of the cut surface of the kidney. But ater writers have applied it to the appearance of the external surface after the capsule has been torn off, as in an advanced stage of this form of disease, the denuded renal surface bears a striking resemblance to a sluggish, granulating ulcer, saving the margins. This use of the term is correct and legitimate, and is not calculated to convey a wrong impression. But if we thus denominate the irregular mottlings of tubal nephritis and fatty degeneration, it is difficult to find any standard of comparison. They indicate simply the existence of epithelial disease which may attend any of the forms of renal lesion, and hence are not distinctive. In this sense a kidney in tubal disease may be granular, a contracting organ may be granular, and so may one affected by the amyloid degeneration. Now then if you present to me such a "granular" kidney, it is still to be ascertained

which of the three types of disease it represents. Since we have high authority for applying the name to the denuded exterior of the organ, and it is much more distinctive when thus employed, it would seem desirable to diminish confusion by using the term in this sense alone.

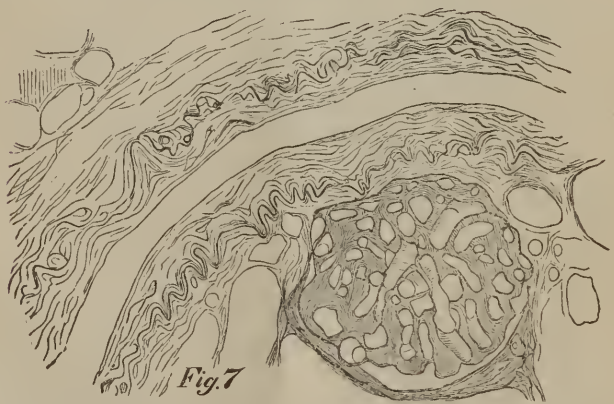
A kidney far advanced in disease of this type, from a person of medium stature, and beyond middle age, furnished the sections from which the next three illustrations were taken. It weighed about two ounces, was tough, firm, somewhat congested, very rough and granular superficially, with a strongly adherent capsule, and a very thin cortical substance, the bases of the pyramids in some places reaching almost to the surface. After appropriate treatment, sections were mounted in balsam and examined.



Granular Kidney, showing increase of stroma. 200 diameters.

Figure six represents a portion of the cortex in the immediate neighborhood of a malpighian corpuscle, whose capsule is somewhat thickened. The vessels near by are also much diseased. One of them appears in the next cut. The noticeable feature here is great hypertrophy of the

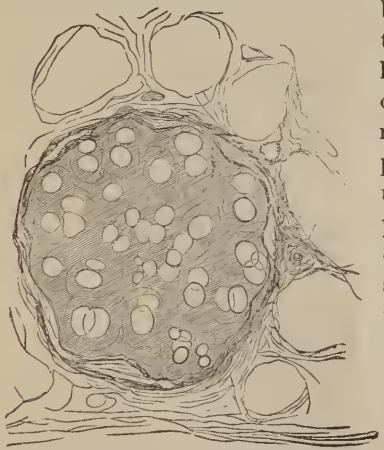
connective tissue, or stroma, at the expense of the epithelial, or glandular element. The tubules are seen cut at various angles of obliquity, contracted, angular, unequal in calibre, and totally devoid of cells. Casts even are absent. This excessive degree of change is not found throughout the section, but here and there in tracts of greater or less area. Near to this portion was found a district where all the tubules were considerably distended; the epithelium, though present, was detached. As far as tubal disease is concerned, the portion represented in this cut had advanced beyond even the stage of fatty degeneration, since nothing is left within the tubes which is amenable to the laws governing that process. The arterial tuft which is partly outlined, is one of those which have resisted the tendency to contraction; it is of about normal dimensions. Many of its neighbors, particularly those adjacent to the renal capsule, were reduced to mere fibrous balls.



Granular degeneration; change in arteries and malpighian tufts. 200 diameters.

By moving the slide a little, a much thickened vessel and a remarkably changed capillary tuft are brought into

view. The arterial walls are seen to be immensely thickened, while the calibre is small and somewhat uneven. A little removed from the canal, the longitudinal fibres are thrown into waves, and upon the outer verge are even plaited. In another specimen might be shown a still more striking instance of this contraction of the blood channels. It is not strange that granular kidneys transmit on an average but one fourth the amount of water through their arteries, in a given time, than will pass through healthy organs.* The diminution of calibre is not caused by a new formation deposited upon the inner walls of the vessel, such as occurs in waxy kidneys. The tissue is seen to be fully formed fibrous material down to the boundaries of the canal. The malpighian tuft in this view is well worthy of attention. It has about the normal size, but is seen to be far from healthy. Transverse and longitudinal sections of variously sized capillary vessels are visible, with an abundance of fibrous tissue between them, making up the bulk of the part. No longer the graceful, nucleated, pliant tuft of minute loops, it is a mass perforated with angular, uneven canals, devoid apparently of cell life. Undoubtedly during the early stages of disease this corpuscle was increased in size, so that even in its later contracted state it is pervious and large.



Granular degeneration. Malpighian corpuscle. 200 diameters.

This same species of change may be carried

* Dickinson Path. and Treatment of Albuminuria, p. 105.

still further, until contraction of some parts and expansion of others have produced the appearance of a homogeneous surface with clean-cut apertures, round or oval in shape, comparatively few in number, and distributed for the most part in pairs. It is scarcely possible to trace a resemblance between the diseased corpuscles of figures 7 and 8, and that of figure 1, which is nearly or quite healthy. The distribution of the openings in pairs, with one, as it often happens, a little out of focus, while the other is sharply defined, is suggestive of their being arranged in loops, and is an illustration of the way in which pathology often demonstrates knotty points in anatomy. In a congested kidney, the minute vascular distribution is often finely shown by the blood corpuscles; obstruction, with either dilatation or withering of a tubule and the attached capsule, will, by contrast with surrounding structures, occasionally exhibit the relation subsisting between them; casts not infrequently demonstrate the anastomosis of the straight tubes; and plugging with epithelium or debris, the looped tubes of Henle. Not to multiply instances, it was a pathological process which demonstrated, after many years of doubt, the relation between the liver cell and the biliary duct.*

Waxy degeneration is chronic in its course, it has no acute stage, and it often invades several regions of the body simultaneously. The liver is most frequently the seat of this morbid process, and the kidneys rank next. Without tarrying to enter debated ground in discussing the causes and nature of waxy change, although these are questions of very great interest, we shall content ourselves with tracing briefly the effects it produces when localized in the kidneys. It is through these organs that its presence is generally made known; although under some circumstances we may be led to suspect its existence in various regions during life, when

* Stiles, Report Met. Board Health, 1868, p. 303.

the kidneys are not invaded. These organs are at first somewhat enlarged, firmer than normal, and allow their capsules to be readily removed. Upon section, they may be somewhat pale, although this is not a constant feature; the malpighian corpuscles appear as grayish translucent points, which reflect light better than the surrounding tissues, and perhaps are larger than normal. During a later stage, the new material, which is thought to be allied to fibrin, undergoes contraction in the same manner as the morbid formation in granular disease. The cut surfaces are then smooth, sharp in outline, glistening, light-colored, and mottled, by reason of the abundance of the so-called waxy points which have increased in size, and become closely aggregated in certain parts near the cortex. In this advanced stage the capsule is often adherent, the denuded surface is sometimes variously excavated, and commonly resembles the surface of a pale granulating ulcer or wound. It is not difficult to detect such an advanced stage by the eye, but this alone should never be relied upon. We have in iodine properly used, an infallible and easily applied test for the presence of this material. Lugol's solution diluted until of a dark sherry color, is a very convenient form. It may be poured over the suspected tissue, or applied by a clean brush and allowed to remain upon the surface for a few seconds, when, if waxy material is present, the affected parts will rapidly absorb the iodine and take a dark brown tint, easily distinguishable from the yellow stain upon tissues not thus diseased. A bottle containing this reagent should be at hand during every autopsy when any form of Bright's disease is suspected, and a minute of the result of its application should invariably appear in the records preserved. Neglect of this precaution seriously detracts from the value of many cases otherwise admirably reported. It is not uncommon to read accounts of obscure forms of disease which after

death afford but little evidence of change to the eye, in which even the microscope has failed to detect serious alteration, when if iodine had been applied, waxy degeneration might have been revealed. In an early stage waxy organs are little altered, and the microscope then renders no such service, unaided, as it does in the other forms of renal disease. There is, indeed, a certain degree of translucency imparted to the capillary tufts and the minute arterial branches (the parts earliest invaded) and the epithelium of the tubules often has something of the same appearance, the cell boundaries being indistinct or altogether lost, but these are indefinite features not easily detected. Again, when the disease is advanced, contraction produces alterations in many respects similar to those which mark the progress of granular degeneration, and there can be no doubt that in the absence of the proper test, waxy organs have often passed as simply granular. This is one of the ways in which the advance of pathology has been much retarded.

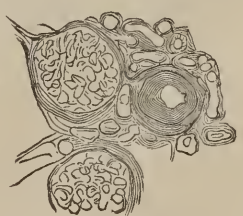
But iodine should be used not alone when renal disease is suspected. We have seen that other organs are subject to this morbid alteration. After any of those debilitating chronic conditions which are thought to induce waxy infiltration, this reagent should be applied to the liver, kidneys, spleen, mucous membrane of the intestines, lymphatic glands, and other suspected parts. Every case of phthisis. of eroding cancer, of syphilis, of caries, of chronic diarrhœa or dysentery, or of other disease, or injury attended with profuse purulent discharge, would be appropriately examined in this manner. In recording observations upon such cases, it is as important to note a negative as a positive result, since in this way alone can a subsequent collator be assured that the reagent has been employed, and be aided to a conclusion as to the proportion of instances of each disease which have suffered waxy degeneration.

The microscope, assisted by iodine, is of the greatest service in detecting early stages of this disorder. And if a section from a kidney in an advanced stage be taken, after being dipped in the solution, the observer will be surprised, if it is his first attempt, at the richness and variety of the tints imparted. The minute arteries thickened and contorted, the malpighian corpuscles comparatively unchanged, and the sessile epithelium, vie with each other to instruct and please. A low power, either the two inch or one and a half inch objective, is preferable to obtain these general effects.

The epithelium after a time is thrown off as in other forms of renal disease. This may proceed to such an extenas to occlude the tubes and light up symptoms of tubal inflammation. Fatty change also may appear, and when this condition is superadded to contraction of the waxy materials, we have apparently all three forms of Bright's disease present in one organ. The casts of waxy disease are apt to be composed either of small fragments of epithelium, or of finely broken casts cemented together, or of homogeneous translucent cylinders often of very considerable diameter. The first two classes constitute the characteristic coarsely granular casts of this type. It is frequently quite difficult to form a diagnosis by the renal derivatives alone.

The peculiarities of this disease which can be made apparent in a wood-cut are comparatively few. We shall repre-

Waxy degeneration.



New Formation in arteries. 40 diameters.

sent one of the most striking, a deposit upon the internal coating of the arteries.

By reference to figure seven, this contraction of the vascular calibre is seen to be different from that which occurs in the granular kidney. The original fibrous and muscular coats are not hypertrophied to any noticeable extent, but there is an opaque, amorphous formation irregularly deposited upon the lining membrane, giving the lumen an angular outline. In prepared specimens this material cut off the light so effectually that it could not be observed with a high power. The deposit in the arteries and the contracted canals of advanced stages account for the diminished secretion of urine often observed in waxy degeneration.

We have now examined the main features of the three morbid processes classed under the title Bright's disease. It has been shown that tubal disease is an accompaniment of two forms, and the essence of the other; that in tubal nephritis, disease is confined to the tubes and their epithelium; in granular disease, primarily to the connective tissue, but that subsequently, tubal disease may appear; in waxy degeneration, primarily to the arterioles and capillary tufts, but that as a consequence tubal disturbance often manifests itself. Contraction and a surface covered, as it were, with granulations are found to be characteristic of advanced granular disease, but appear also in waxy degeneration from similar causes. Fatty degeneration is reduced from the rank of a disease to that of a symptom; consequent upon sustained epithelial disturbance, it appears whenever this condition obtains.

The "large white kidney" is a result of tubal nephritis, which in its chronic stage has resolved itself into fatty degeneration. The "small white kidney" may be a result of either granular or waxy change. Such are the combinations, and the successions generally observed. Waxy infiltration

is said, however, to follow occasionally in the train of tubal nephritis.

In conclusion we may allude to the sometimes difficult matter of determining the original lesion as we examine combined forms of kidney disease. We have seen that Stewart and others constitute a third or contracting stage of tubal nephritis. But such a state of the organs must be brought about by two processes, contraction of the fibrous element, and disease of the tubes. Which of these has caused the other cannot be determined by the microscope alone. It is a question for clinical observation to decide.

Of itself tubal nephritis is not known to cause, or, in any great proportion of instances, to be followed by hypertrophy of the left ventricle of the heart, or valvular disease of the same organ, or cirrhosis of the liver. On the other hand these are the commonest attendants of granular disease. Hence, if we meet the combined form above mentioned with such concomitants, it is quite certain that the granular disease is the older lesion. So in waxy degeneration, if a contracted kidney is associated with a considerable infiltration of other organs, known to be liable to such change, it is not difficult to decide that any tubal disturbance discovered must be a result and not a cause of the renal lesion. Again, if it should be questioned whether the contraction of such a kidney were not due to granular degeneration, clinical observation would show that a large liver, a heart almost always free from chronic disease, and the presence of some exhaustive drain upon the blood, conditions not found with the granular kidney, point to a process of disease essentially different.

In Bright's disease, then, there are included three types of morbid change, but in accordance with certain well-defined laws, these may be variously combined in the same organ.

THE

PATHOLOGY

OF

BRIGHT'S DISEASE,

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